Savill Oration

ON

THE TRAUMATIC FACTOR IN ORGANIC NERVOUS DISEASE*

BY

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The question as to the causal relationship between injuries to the head, back, trunk, or limbs and the subsequent development or aggravation of a pre-existing organic nervous disease has been much debated, and entirely contrary opinions have been expressed by various authors. In my opinion grave injustice is often done by the refusal of consultants to allow the possibility of cause and effect in progressive nervous disease following severe blows upon the head or spine. It will probably be conceded by all competent observers that epilepsy may be a direct sequel of, and dependent upon, an injury to the skull, whether this is a severe blow, a fractured skull, or a perforating wound such as a rifle bullet or shell wound. The injury to the brain in these three groups of cases is in order of increasing severity, and the actual injury to the nervous tissue will not be a matter of dispute in cases of fractured skull or perforating wound.

It is the first group of blows on the head of less severity, yet followed by recurrent epileptic fits, that may give cause for various opinions. The nature and severity of the blow and the interval before the first development of symptoms must be taken into account, together with other factors, such as heredity of epilepsy, fits in earlier life, antecedent syphilis, etc. I have seen in private practice two cases of recurrent epilepsy, which followed directly after a knock-out blow in boxing contests, in previously healthy, normal men. The lesion produced by the blow causing the concussion, or commotio cerebri, may not be demonstrable, but probably consists of numerous small ecchymoses or punctiform haemorrhages, comparable to a bruise on the surface of the brain or beneath.

What effect concussion may have directly upon the cerebral nerve cells, in the absence of haemorrhages, is speculative, but it is not difficult to imagine that the cell's delicate structure may receive some injury affecting its metabolism and function which may be permanent, and may even start progressive changes. A good instance of traumatic epilepsy is the following:

A road sweeper, aged 51, was knocked down in the street by a motor lorry on January 19th last, striking the back of his head on the road, and being unconscious for a few minutes, but the scalp was not cut. *X* rays showed a fracture of the base. A week later he collapsed, with seven fits in succession, and he has had five fits since. He had never had a fit previously in his life, nor were any known in his family.

Such a severe injury with fractured base may be assumed to cause contusion of the brain in one or more places, and adhesions may develop with a vascularized scar, causing the brain to adhere to the dura, and setting up an irritant focus, leading to epileptic fits. How such an injury can set up the multiple progressive lesions found in disseminated sclerosis is difficult to understand. Probably many cases can be explained by the actual disease being already latent; the effect of the injury is then to bring into prominence, and to hasten, the development of the disease.

Such is no doubt the order of events in cases of G.P.I. which develop rapidly after injury to the head or back, the antecedent neurosyphilis being latent, and possibly the nervous degeneration of G.P.I. might never develop if it were not for the immediate effects of the blow. On looking through my private case books I have found several cases of G.P.I. in which trauma appears clearly to have been a factor in the development of the symptoms. The following case is a good example.

A flying officer, aged 42, made a parachute descent in August last year, came down too fast, and bumped his head on the ground rather badly, being partially concussed and dazed, though he carried on with his work. The same night he had a severe headache. During the same week he became sleepy, slept during dinner, and in many ways did not know what he was doing. He slept badly at night, with horrible dreams. A week after his crash he got pains in his arms and legs and back, with the neck stiff, pain lasting for three days only. He reported sick two days later, and it was then observed that he was becoming very forgetful, developing curious ideas, wanting to breed racehorses, and he wanted a divorce from his wife, though they were really a devoted couple. He became very impatient and irritable, careless in his dress, and forgot to shave.

On examination on September 13th, 1932, his right knee-jerk was absent, though the pupils were active; some patchy analgesia of the legs and forearms. His utterance was normal, but the wife stated that his memory was not so good, though he had improved in his mental condition considerably. The cerebro-spinal fluid showed a strong positive Wassermann reaction, 36 cells per cubic millimetre, mainly lymphocytes, the Lange curve showing 555543100. He had been fully treated for syphilis twelve years before.

I treated him with malarial inoculation by mosquitos, sent from the Ministry of Health, and after sixteen days' incubation the temperature began to rise, and he had nine rigors in the next fortnight, the pyrexia then ceasing without quinine being administered. He has since made a steady recovery, though three months later, in December, the Lange test was still 5555543200, but the cells were reduced to 4 per cubic millimetre, and the Wassermann reaction about one-third the strength it had been in September. I have given him no further medicinal treatment, and he has been perfectly fit in every way since, with the exception of a recurrence of malarial pyrexia, and on August 29th, 1933, his cerebro-spinal fluid showed perfectly normal to the four tests; Wassermann negative, Lange 0000000000, and 2 cells per cubic millimetre with normal globulin.

In this patient there had been no mental symptoms before the injury; he was apparently in perfect health until the time of the blow, and it seems impossible to disagree with the view that the injury was the direct exciting cause of the development of the symptoms, though the condition of the cerebro-spinal fluid is clear evidence that the disease was latent. Whether in this or similar cases the disease would never have developed without such an exciting factor as an injury must, of course, always be speculative. No doubt severe worry and mental overstrain may have a similar accelerating effect in the development of general paralysis, of which I have seen more than one example.

If we look up the aetiology of various organic nervous diseases in textbooks of medicine or pathology the influence of trauma is usually scantily treated or not mentioned. In Osler's *Medicine*, edited by McCrae (eleventh edition, 1930, p. 1143), the following interesting, though brief, opinion on the effects of trauma is given:

"In rare cases following trauma and succeeding to symptoms which may have been regarded as neurasthenic or hysterical, there are organic changes which may prove fatal. That this occurs is demonstrated clearly by post-mortem examinations. The features upon which the greatest reliance can be placed as indicating organic change are optic atrophy, bladder symptoms, particularly in combination with tremor, paresis, and exaggerated reflexes." (I might also add the Babinski toe reflex.) "The anatomical changes are not very definite. When death follows spinal concussion within a few days there may be no apparent lesion, but in some instances

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the brain or cord showed punctiform haemorrhage. Edes reported four cases in which degeneration in the pyramidal tracts followed concussion or injury."

Again, in an article on progressive muscular atrophy, by Drs. Collier and Adie, in *A Textbook of the Practice of Medicine by Various Authors*, edited by F. W. Price (second edition, 1926), they say (p. 1579):

"Injury is an important exciting factor, and cases are not very uncommon in which progressive muscular atrophy of typical course immediately followed upon an injury, and commenced first in the region of the injury. We have twice seen this malady commence in the muscles of the back after a blow upon the spine. Commencement in a limb which has recently been injured is more frequent, and general traumatic influences, such as are occasioned by a fall, not very uncommonly precede the onset of symptoms in patients of later years; and the course of the disease in such cases is likely to be rapid."

These authors base their description of the clinical features of the disease upon an analysis of 500 cases which have come under observation at the National Hospital, London. On comparison of this article with their article on the same disease in the third edition of 1929 it is somewhat astounding to find that the whole article from beginning to end is word for word the same in the two editions, with the exception that when speaking of the aetiology of the disease in the latter edition an exactly contrary opinion as regards the effect of injury is expressed. In the 1929 edition they say (p. 1625):

"Injury has often been alleged to be an exciting factor in the development of this disease, and there are very many cases recorded which seem to support this contention. We have made a careful analysis of many cases in which there has been a history of injury which have come under our observation, and also of those in the literature, and have found that it is always a trivial injury, such as a slight knock, bruise, or strain, and never a severe injury, which has preceded the appearance of progressive muscular atrophy. This one fact clearly proves that injury cannot truly be in causal association with the disease, and, further, there is no conceivable pathological process by which a trivial injury, unaccompanied by any infective process, can bring about a universal and rapidly fatal dissolution of the nervous system. The probability is that the injury is in many cases due to the slight clumsiness and disability often produced by the oncoming disease, and that in other cases it is simply co-incidental."

The article in the recently published 1933 edition is also identical. No explanation is given in the last editions for this complete reversal of their opinion as regards the effect of trauma, although the whole article, with the exception of this paragraph on injury, is word for word the same in the two editions. They make no reference whatever to trauma as a factor in the development of disseminated sclerosis or of G.P.I., but their discussion of the subject of trauma in their article on progressive muscular atrophy may probably be taken as their expressed opinion to include other degenerative nervous lesions, and this view is perhaps that held by the majority of neurologists to-day.

To combat this materialistic view is one purpose of my present oration, and I would point out a serious error in the authors' argument, where they state that they have made a careful analysis of many cases with a history of injury which have come under their observation, and also of those in the literature, and have found that it is always a trivial injury, such as a slight knock, bruise, or strain, and never a severe injury, and they go on to state that this one fact clearly proves that injury cannot truly be in causal association with the disease, and, further, there is no conceivable pathological process by which a trivial injury, unaccompanied by any infective process, can bring about a universal and rapidly fatal dissolution of the nervous system. This latter statement is a clear instance of sophistry or special pleading, in assuming that their present knowledge of pathological processes is final, and not to be enlarged by new facts and knowledge in the future, and indeed it shows a certain degree of want of vision.

From the very nature of the case it is impossible to prove whether injury has originated a progressive nervous disease, for it would be necessary to examine the brain and cord before the occurrence of the injury in order to prove that no latent disease was present. It is only by recording numbers of cases which bear upon the point, and considering each case upon its merits, that a careful and unbiased judgement can be reached. The cases which I shall refer to later on will be seen to have suffered serious blows, often causing unconsciousness, and not to be compared with the "slight knock, bruise, or strain" referred to in Collier and Adie's article. The following case of progressive muscular atrophy came under my observation for the first time in June, 1931.

The man was a railway porter, aged 35, in robust health in July, 1930, when he had an accident while wheeling a heavy case weighing 5 cwt., which fell off the barrow on to his right foot, bruising it very severely. No fracture was found on *x*-raying the foot, which was then put up in plaster-of-Paris a fortnight after the injury, for two weeks. From the time of the injury he complained of pain in his foot and up the front of the leg, which ached considerably, and there was weakness of dorsiflexion of the foot from the commencement. The foot was still swollen when the plaster was removed, and was very painful; signs of bruising persisted, and he went about on crutches. To put it shortly, he has never recovered the use of his leg, and it has always been painful, though he did light work at his own request for ten weeks during the winter of 1930. Pes cavus developed and the foot was wrenched under gas, and he then had massage, with a dropped foot, and gradually during the following months the muscles of his leg wasted below the knee and the foot dropped further.

When I saw him first, eleven months after the accident, there was considerable wasting of the dorsiflexors of the foot, with a steppage gait. The cerebro-spinal fluid was normal to all four tests, fibrillary tremors developed during the following winter, and in April, 1932, there were fibrillary tremors in both triceps muscles and in the extensors of the wrists and fingers.

In October, 1932, the pains were increasing so that he was unable to walk more than a few yards without pain in the legs, beginning in the right thigh, down the front of the leg, and across the sole, with sharp, stabbing pains which had begun a month after the operation for pes cavus. The right ankle was completely dropped, and extension of the ankle was weak. The extensors of the right thigh were also weak, and the left anterior tibial muscles were even more wasted than those on the right side; there was extensor plantar reflex on the left side, with flexor on the right, or injured, side. There were no bulbar symptoms, the conditions remaining much the same in the spring of this year.

It is evident that the man is developing amyotrophic lateral sclerosis, and the immediate onset of symptoms in the neighbourhood of a very severe injury to his foot is extremely suggestive of causal relationship between the injury and the subsequent disease. When the case was tried at the Middlesex Sessions, however, the man lost his case on a technicality—namely, that the medical referee in the country had previously given his decision that the injury had nothing whatever to do with the disease, without having heard any special evidence on the man's behalf. Such is the law, that that verdict was final, and that that point could not be reopened. Verily, as Bumble remarked, "The law is a ass."

What can be the causal connexion, if any, between such an injury and the subsequent disease? We know that injury to peripheral neurons produces chromolytic changes in the nerve cells in the spinal cord or posterior root ganglion connected with the peripheral nerve fibre that is injured, a metabolic change which can be recog-

nized microscopically, and is termed the "réaction à distance" by Marinesco. As that is indisputable it is conceivable that under certain circumstances further changes of a progressive nature may result, and as at the present time we know nothing whatever in reality of the pathology of progressive muscular atrophy, what right has any author to take up the point of view that there is no conceivable pathological process by which an injury, unaccompanied by any infective process, can bring about a universal and rapidly fatal dissolution of the nervous system?

Until we know more, and have accurate knowledge of the pathological processes underlying such diseases as progressive muscular atrophy and disseminated sclerosis, we have no right to deny the aetiological factor of trauma when the history of the case clearly shows a close time relation between the injury and the onset of the symptoms. During the war I saw a case of pituitary tumour develop directly after a man was injured in a bombing accident when at practice. Following the injury to his head, he suffered from continual headaches, later developing typical bitemporal hemianopic scotomata from the pituitary pressure, an adenoma of the pituitary being demonstrated by operation. Can this be passed off as pure coincidence, or was the injury partly responsible? My own view is that when symptoms develop directly after an injury in a previously healthy individual we have no right whatever to assume that the injury has no causal connexion with the development of the disease. In such case we should be assuming a supernatural knowledge, which is not yet vouchsafed even to the writers of articles in textbooks.

Cerebral Tumour

A history of previous injury is very frequent in cases of cerebral tumour, and it is always very difficult to decide what the connexion can be, if any. We have to consider the severity of the injury, the health of the individual at the time, and the lapse of time before the first development of symptoms. Indeed, Drs. Collier and Adie, in their article on cerebral tumour, say (Price's *Medicine*, fourth edition, p. 1496):

"The relation between cephalic trauma and the first appearance of cerebral tumour is one which occurs much too often to be ignored, though it is likely, in many of the cases in which this relation exists, that the blow on the head has simply served to bring a pre-existing tumour into symptomatic prominence, either by causing oedema or haemorrhage in its substance or vicinity."

If in a healthy individual symptoms follow very shortly after an injury of some severity I should certainly be inclined to allow that the trauma had so affected the tissues of the brain as either to hasten the onset of symptoms due to a latent tumour by setting up haemorrhage, or oedema around it, or possibly even through some metabolic process, of which we have no knowledge, that the injury may even start the formation and development of tumour cells. The former alternative of injury hastening the development of symptoms hitherto latent will probably be accepted by most observers; the latter suggestion that tumour cells may arise *de novo* as the result of an injury is a field for entirely speculative argument, though it should be observed that we know that in many instances tumours such as scirrhus of the breast or epithelioma of the lip or tongue and elsewhere may arise as the direct result of local injury.

Disseminated Sclerosis

On analysing my private cases of disseminated sclerosis—a total of 234, seen in the last twenty-eight years—I have found sixteen cases in which definite chronic spinal sclerosis, mostly cases of typical disseminated sclerosis, has developed shortly after severe injuries to the back or head, several of whom were concussed and unconscious for a time varying from several minutes to several days. In two of these cases paroxysmal neuralgic trigeminal tic was a complication in each instance, antedating the symptoms of spinal sclerosis.

This group of 234 cases is, I consider, moderately certain as regards the diagnosis, because I have eliminated all cases in which there seemed to be a reasonable doubt. All these sixteen gave a strong history of injury—that is, in 7 per cent. of all my cases. I will now give the details of the more striking of these cases, as trauma has not, in English literature, usually been considered as a factor in the development of this disease, though many foreign writers have laid stress upon the aetiological factor of trauma. Oppenheim, in his textbook on nervous diseases, quotes many other German writers on this point.

Case 1.—A man, aged 30, was wounded in 1916 in France by a rifle bullet, which pierced his right shoulder, coming out on the left side, and causing paralysis of his legs and arms. The left arm and leg have never completely recovered, but he made a fair recovery, though the legs were never completely strong again, though he was able to return to France. In January, 1918, he was again wounded in the right shoulder, by a shell, and he was gassed in the summer of that year. He was demobilized in 1919. His legs have been weak ever since, and his arms again began to lose power. Ever since the original injury in 1916 he has had weakness of sphincter control. He has gradually been getting weaker, both in arms and legs, since he was demobilized. Two and a half years ago he began to develop paroxysmal neuralgic tic of the right second and third divisions of the fifth nerve, with typical spasms on eating, talking, or handling the face. When I saw him last June he had an ataxic, spastic gait, with slightly staccato speech, ataxic nystagmus, and pallor of the right disk; the left arm and leg were weak, the left plantar extensor in type. I injected his Gasserian ganglion to arrest the neuralgia, but the progress of the spinal sclerosis is likely to be continuous.

The diagnosis of disseminated sclerosis can hardly here be disputed, in view of the staccato speech, optic atrophy, nystagmus, and the spastic ataxy; and, moreover, the development of paroxysmal neuralgic tic, as I have shown elsewhere, is peculiar to disseminated sclerosis. Yet his paralysis dates from the actual bullet injury to the spinal cord, and the weakness has been more or less progressive steadily since. How is it possible to deny in such a case the aetiological connexion between the original injury and the subsequent organic nervous disease?

We have no idea what is the pathological process that produces the nervous degeneration and islets of sclerosis in the brain and cord in this disease. That injury had some direct causal connexion in this case I think must be agreed, but whether it accentuated a process already latent or whether it had started the disease *de novo* is entirely speculative.

Case 2.—A young man, aged 21, had a bad fall from a motor bicycle, damaging his right hip and shoulder. Within the same month he noticed "pins and needles" down both arms to his fingers on bending his neck forwards, making him giddy and nauseated. This symptom has been many times observed in the early history of disseminated sclerosis. After a year he became better, and eighteen months later, when writing, he noticed numbness of the inner two fingers on his right hand, which has never quite recovered, and spread next day to the pads of the outer two fingers, his doctors telling him he was suffering from hysteria.

The following year he was involved in two taxi accidents in Paris within five days, a man being killed in the first accident, and within a week or two he became staggery when he tried to run. Spinal tumour was suspected also by a neurologist at that time, but he improved considerably after some months. When I saw him in 1926 he was ataxic, with spastic physical signs, numbness and partial anaesthesia of

the inner left forearm, and blurred vision, with sphincter weakness.

Although this cannot be considered a demonstrable case of disseminated sclerosis, yet it is clearly one of organic nervous disease, which showed its first symptoms within a month after severe injury.

Case 3.—A woman, aged 49, seen by me in April, 1924, had had a severe fall six months previously, striking the back of her head on a lavatory basin. Ever since she had partial loss of control of both sphincters, with precipitancy. During the last six weeks she began to develop numbness and pins and needles on the left side of the breast down to the level of the foot, the left leg was weak, the left foot feeling cold, and it slightly dragged on walking. No affection of the arms. The knee-jerks were increased, the left greater than the right, the left plantar extensor, double ankle-clonus, abdominal reflexes absent, and slight sensory changes of hyperaesthesia to pin scratch on the left side of the leg and abdomen up to the eighth dorsal level. No nystagmus or diplopia.

As regards diagnosis, the immediate effect of the injury was to produce partial sphincter paralysis, and four and a half months later gradual progress of spastic weakness of the leg.

Disseminated sclerosis certainly seems the most likely explanation, although she was perfectly well before the injury.

Case 4.—Another war case was a man of 33, whom I saw in December, 1930. When aged 21 he crashed from a motor bicycle when carrying dispatches in France in May, 1918, falling forwards and, as he said, skidding on his chin. When he picked himself up he found that his left ankle was weak, and he has never recovered the strength of that leg, always dragging the foot. It was not until 1926, eight years after his injury, that his left hand began to be numb and weak. For the past eighteen months he has had bladder incontinence. The plantar reflexes were normal, and there was no increase of the deep reflexes. The abdominal reflexes were absent, and he had well-marked ataxic nystagmus of the right eye particularly. Three months after I saw him his right side became affected, with considerable sensory defect to changes of temperature, not readily discerning hot from cold.

The absent abdominal reflexes, with loss of sphincter control, and ataxic nystagmus coming on very gradually, are very suggestive of disseminated sclerosis, and the fact that the weakness of the leg dated from the actual injury in France is certainly suggestive of a severe blow upon the head having damaged the nervous system and started some progressive nervous degeneration, which probably is disseminated sclerosis.

Case 5.—Another war injury was in a woman, aged 60, whom I saw in June, 1931. Fourteen years previously, when she was aged 46, in December, 1917, her house in Clapham was struck by a bomb, and she was thrown violently across the room. Her husband at the same time was blown violently about, became epileptic, and died shortly afterwards. She began to get shaky, and soon became weak in walking, and the weakness has slowly increased since then. She is liable to attacks of trembling and inability to speak. On examination in June, 1931, she had a weak ataxic gait, though no ataxy of the arms. The knee-jerks and deep reflexes were normal, but the plantar reflexes were bilaterally extensor. There was some tremor of the body and head, and she was subject to emotional attacks. There was no nystagmus, and she was able to knit and to use her arms, but since then she has developed epileptic fits.

The violence of this injury is apparent, and without doubt the brain was contused and probably numerous punctiform haemorrhages in the cortex and meninges resulted, but the gradual development soon afterwards of shakiness and weakness of gait, later with ataxy and extensor plantars, and finally epilepsy, is an incontrovertible indication of the development of a chronic nervous disease as the sequel to the brain injury. Possibly this chronic sclerosis is of the disseminated type.

Case 6.—A girl of 22 was sitting at lunch in a restaurant in the City when a heavy ventilating propeller, with its attached motor, fell from the ceiling directly upon her head, causing her to fall forwards, eight months before I saw her in October, 1919. She was given brandy and encouraged to eat, but she was unable to do so. She felt ill, and left the restaurant and returned home. She felt sick after the blow, and on reaching home her head felt heavy and the neck ached; she was kept in bed for three days, and kept away from work for six weeks. She complained continually of headaches and pain in the neck, back, and head, nervousness, and crying attacks, with bad dreams at night, when she felt she was falling. She slept badly, and often had to go to bed on account of headaches and pain in the back. Her mind felt confused and muddled, her memory was bad, and she forgot appointments. She would shake and scream, and might jump out of bed in her sleep. She tired easily, complained of a constant funny feeling in the head "as though a wind were blowing on the brain." Her doctor told me that both she and her people were rather casual about the accident, and did not take it at first seriously.

When examined in April her knee-jerks were found to be exaggerated and her hands tremulous, the plantar reflexes being flexor. When I saw her in October, eight months after the accident, she said she felt giddy if she tried to dance, her knees giving way, and she had to stop at once. The pain in the head soon after the accident became limited to the left side, but she still screamed at night and slept badly, and she broke down crying in my room. On examination the knee-jerks were exaggerated, there was no weakness of the feet or legs, and no anaesthesia. The left plantar reflex was constantly extensor, and the right occasionally so. When I saw her a month later she then had constant bilateral extensor plantar reflexes, with absent abdominal reflexes, which fact was confirmed by Dr. James Collier, who saw her with me.

This is a clear case of a severe blow upon the head causing concussion, followed immediately by headaches and pains, and emotional attacks, bad dreams, etc., which are commonly associated with a diagnosis of neurasthenia; yet the absence of the abdominal reflexes, and the later development of bilateral extensor plantar reflexes, must be taken as incontrovertible evidence of progressive nervous degeneration, which very possibly may be disseminated sclerosis.

Case 7.—A hospital nurse, aged 26, fell heavily down some stone steps, becoming unconscious for half an hour. She was warded for two months on account of pain in the back, during which time it was noticed that she dropped things and limped on the right leg. Eighteen months later she left the hospital, still limping on the right leg, and she has never been well since, complaining of a dull ache in the back and dragging sensation with numbness of the right leg, and weakness on going upstairs. Her trouble at first was mistaken for hysteria.

When I saw her in 1916, eight years after her injury, she had an ataxic gait and weakness of the right foot, the knee and Achilles jerks were exaggerated, and bilateral extensor plantar reflexes were present. There was slight ataxy of both hands, especially the right, and there was bilateral optic atrophy. Bladder incontinence was present, and there was loss of vibration on the lower extremities and on the abdomen up to the fifth dorsal level.

Here again severe injury was followed at once by pains in the back, weakness of the arms and leg, and later development of definite symptoms of disseminated sclerosis. Are we to assume that here was a case of early disseminated sclerosis unrecognized, but, owing to her disease, she falls heavily, and injures her spine? In any case the symptoms were aggravated by the fall, and there is no reason to suggest that she was anything but perfectly healthy and normal before the injury. I cannot but believe that the development of disseminated sclerosis was directly due to the severe blow on her head and back, which caused severe concussion.

Case 8.—An unmarried lady, aged 51, was run into by a car in January, 1923, the wing of the car hitting her left shoulder, and knocking her down on to the ground, striking her right shoulder heavily. No bone was broken, and she picked herself up and was then taken home. The right arm was weak from the commencement, and she was unable to hold anything firmly, and would drop her cup and other articles. After some weeks the arms gradually recovered. Six months later she noticed that her legs seemed to drag, especially the right leg. When I saw her in October of the following year both knee-jerks were much exaggerated, the right greater than the left, with bilateral extensor plantar reflexes and right ankle-clonus, with loss of vibration sense on the right leg, but no other sensory symptoms, no sphincter weakness, and eyes normal.

Here again a woman in perfect health after a severe injury has temporary weakness of the arms, and six months later gradually progressing weakness of the legs of spastic type. Organic disease was certainly present, and its connexion with the injury is difficult to exclude. Disseminated sclerosis seems a strong probability.

Case 9.—A boy of 15 had a severe crash from a motor bicycle, and was unconscious for a short time. He was not allowed to play games for a time after his return to Clifton, but he afterwards played rackets for his school. Two and a half years after the accident he developed diplopia and during the last year weakness of his legs. When I saw him in April, 1924, he had an ataxic gait, bilateral extensor plantars, ataxy of the left hand, absence of the left abdominal reflex, slow scanning articulation, and marked nystagmus, both lateral and vertical. He died the following year, but no necropsy was permitted.

Diagnosis of disseminated sclerosis seems quite clear in this case, and though there is a history of severe concussion two and a half years before the first symptom of diplopia developed, the length of time is too great to assume a necessary connexion between the trauma and the subsequent disease, though the fact is suggestive when taken in conjunction with the numerous other cases of trauma preceding nervous disease.

Case 10.—A woman, aged 53, was badly concussed in a motor accident, being unconscious for a time. A fortnight later paroxysmal neuralgic tic of the left third division of the fifth nerve commenced. She had no doubt suffered from disseminated sclerosis for thirty-five years, and when I saw her in 1928 she was limping on the left leg, walking with a stick, with flaccid paralysis of the left ankle, and bilateral extensor plantars. She was sent to me on account of the trigeminal neuralgia, which was arrested by alcohol injection.

In this case it would appear that the severe injury was concerned in the starting of the trigeminal neuralgia in a case of chronic disseminated sclerosis. I have published elsewhere[1] a case in which paroxysmal trigeminal neuralgia commenced within an hour and a half after a severe blow on the chin by another person's head, and I have similarly seen a blow on the jaw or face set up trigeminal neuralgia in a few other cases.

Case 11.—A man, aged 20, was badly buried in 1917 by a shell explosion at Ypres, being unconscious for eight weeks. He was then practically blind, could see shadows only, could not control his speech from stammering, and was extremely nervous. Eleven years later his left leg suddenly collapsed, and numbness spread up the left side of his body. When I saw him in 1930 there was right optic atrophy, with difficulty in moving his eyes in any given direction, bilateral ankle-clonus, left extensor plantar, and numbness of the left leg and arm. He said that the left side had never really recovered since the shell explosion and burial in 1917.

This is another case of a war injury directly producing symptoms of nervous disease which have never completely recovered, and, in addition, later symptoms of early disseminated sclerosis have developed. It is probable that a person without prejudice would connect the whole story as one, and even consider that the disseminated sclerosis is entirely due to the early war injury.

Case 12.—Another suggestive case was that of a lady, aged 47, whom I saw in October, 1927. Six years previously, when aged 41, she had a bad fall on a polished oak floor, sitting down very heavily and knocking her spine with a violent blow against a step. After this she felt pain on laughing, with a sensation as though she were cased in iron. A year later she began to suffer from giddy turns, and gradually her walking became weak, and she was apt to fall, especially the right foot being liable to drag. Some sphincter weakness of the bladder developed, but that is now better. Her articulation is slurring and staccato in type, the plantar reflexes are bilaterally extensor, and the knee-jerks are exaggerated. The arms were normal, and there was no nystagmus or other signs of disseminated sclerosis.

In this case there was an immediate onset of symptoms following the injury, though it was not for another year that definite symptoms of progressive nervous disease developed. I think it would be right to assume that the injury had some connexion with the development of her symptoms, either in aggravating previous latent spinal disease or possibly originating it.

Case 13.—Another war case was that of a man who, when aged 25, in September, 1915, fell heavily off his horse when jumping a pole, and struck his upper dorsal spine on the ground out of doors, and an hour or two later he complained of pins and needles in both forearms, especially in the hands and fingers, and he could not bear to touch anything for three weeks. He was dazed by the fall and could not pick himself up, and was put to bed for six weeks. The hands were weak at first, as well as suffering from paraesthesiae. His memory since the accident has been bad. His legs were weak, and he could not walk more than a mile. Gradually he improved, and returned to take up his commission, on light duty, with a cavalry regiment. Though he twice fell, his legs were weak in gripping his horse, and his arms recovered completely. In December, 1918, three years after the injury, he noticed his right vision failing, which he is certain must have developed in the previous six weeks. The following April his left eye began to fail, and when I saw him in December, 1919, he had bilateral optic atrophy, with almost complete central scotomata, bilateral extensor plantars, though no ataxy or nystagmus. The injury produced by the fall clearly concussed his spinal cord, as evidenced by the pins and needles in the forearms and hands an hour or so later.

This man was previously perfectly healthy and normal, though his legs afterwards became weak and his plantar reflexes extensor, with development of optic atrophy, the symptoms being suggestive of disseminated sclerosis. A radiograph taken in the following year is said to have shown a fracture of the right transverse process of the seventh cervical vertebra. The actual lesion of the spinal concussion may have probably caused meningeal haemorrhages and diffuse small haemorrhages in the spinal cord, though three years later he developed subacute retrobulbar neuritis, with subsequent optic atrophy.

Case 14.—A man, aged 50, had a bad fall from a motor cycle in 1918, heavily bruising the right leg, which had become gradually weaker since the time of the injury, and he has dragged his right leg ever since. Two years before his injury he was suddenly struck with violent paroxysmal neuralgia of the right third division of the fifth nerve, the pain being very violent, like hot electric wires in his lower jaw. Since then the neuralgia has attacked him from time to time, though he had an interval of freedom of twelve months at first. When I saw him in 1922 he had a typical spastic right leg, with extensor plantar reflex, some conjugate weakness of the movement of the eyes to the right, with slight nystagmus. I injected his third division with alcohol, causing total anaesthesia of its distribution, and completely relieved the pain. Three years later, in 1925, his gait was more spastic and the right thigh wasted, with marked fibrillary tremors of the extensors, bilateral extensor plantars, and some ataxic nystagmus.

In this case the weakness and progressive spasticity of the right leg dated directly from the motor cycle fall,

though later on the spasticity increased, the plantars became bilaterally extensor, with some ataxic nystagmus and some wasting of the extensors in the thigh. The injury appears to have produced some damage, possibly in the spinal cord, damaging his right leg, but beyond that there is a progressive element of nervous disease as shown by the nystagmus and the fibrillary tremor and wasting of the extensor muscles in the right thigh. The absence of anaesthesia and sphincter symptoms might suggest, in view of the wasting and fibrillary tremors, amyotrophic lateral sclerosis, though the ataxic nystagmus is more in favour of disseminated sclerosis. In any case, a progressive nervous disease following immediately upon severe concussion of the spine is evident. A point in favour of the disease being really disseminated sclerosis may be taken to be the presence of trigeminal neuralgia, and, seeing that this had commenced two years before his injury, it is quite possible, if not probable, that disseminated sclerosis was lying latent and was aggravated definitely by the injury.

Case 15.—An interesting case because of its early commencement was that of a man whom I saw in 1920, aged nearly 23. When 8 years old, for three months he suffered with incoordination of both his hands and loss of sensation. He never walked well in the dark, but he was a fair long-distance runner as a boy. Diplopia was first observed when he was 15 years old, but he had no weakness of his legs until three months before I saw him, and five months previously to commencement of the weakness of his legs he had had a bad fall from a horse, and shortly before that a severe blow on his head.

On examination he was a typical case of disseminated sclerosis, ataxic, with bilateral extensor plantars, bladder incontinence, and nystagmus, especially to the right. It seems probable that the two severe blows that he had a few months before the onset of the weakness of the legs aggravated the existing disease and caused it to progress more rapidly.

Case 16.—Another case of trigeminal neuralgia complicating chronic spinal sclerosis I saw in a man, aged 49, who had had a severe fall when bicycle racing, being badly concussed and unconscious for several days. He was laid up for some weeks after this, and his right leg never became quite right again. Five years later he had a fall when out shooting, causing numbness of the legs, which were paralysed for an hour.

The case was mistaken for a pressure paraplegia as a result of the fall, and laminectomy was done, and seven years later trigeminal neuralgia commenced. When I saw him he was very spastic and ataxic, with extensor plantars and absence of abdominal reflexes. The case appears clearly to be one of a chronic spinal sclerosis, possibly disseminated sclerosis, developing as a sequel of severe spinal and cerebral concussions.

Case 17.—A case I saw at St. Mary's in 1919—a man of 36, who, when aged 22, was struck on the head by a falling log of wood, and was unconscious for a quarter of an hour. From then onwards he complained of sensations of weight on the top of his head, though he kept at work for eighteen months. Then one night he had a "stroke," his left arm and leg being paralysed for weeks, and his face drawn over. In two months' time he was better, and was able to return to his work for three months at the time, when the weakness would return, either on the right side or left, accompanied by sphincter trouble. On examination I found spastic paraplegia, with some ataxy of his hands, well-marked nystagmus, extensor plantar reflexes, and ankle-clonus.

This case was clearly one of disseminated sclerosis, which appears to have had its origin in the blow upon the head three years previously. In addition to these cases I have notes of six more hospital cases in which the symptoms in disseminated sclerosis appeared to follow directly after injury, but it is, I think, unnecessary further to labour this point.

Owing to the greater liability of the brain to contusion from injury it is not a matter of surprise to me that cerebro-spinal sclerosis appears to be a far commoner sequel of injury than is tabes or progressive muscular atrophy—degenerative processes almost limited to the spinal cord. The spinal cord is far more carefully protected from injury than is the brain. Hanging suspended by ligaments in a water-jacket contained within a jointed bony tube, which is itself encased in thick musculature, it is not surprising that it is able to withstand moderately severe blows upon the back without gross injury. Severe falls or violent blows can no doubt effect direct damage to the spinal cord itself through all its defences, sometimes by fracturing a vertebra and causing direct contusion of the cord, which may lead to transverse myelitis. Such fractures of a vertebra are to-day more easily demonstrated by improved technique in taking lateral *x*-ray photographs of the spine. Kümmell's disease has thus been shown to be definitely a fracture of the body of the vertebra and not a shock absorption of the bony tissue due to a blow.

Concussion of the cord, without fracture, sometimes produces immediate symptoms of pins and needles in the arms and hands, hyperaesthesia, and curious sensations like electric shocks on flexing the head. Almost certainly punctiform haemorrhages are produced in this way, the immediate symptoms being aggravated a few days later by oedema secondary to the haemorrhages and contusion. Nerve cells may thus be damaged and then atrophy; and who in our present state of pathological ignorance, I will not say knowledge, can confidently assert that such a process, once started, may not progress, even to a fatal termination?

REFERENCE

[1] *Neuritis and Neuralgia,* Oxford Medical Press, 1926, p. 169.

THE TREATMENT OF TUBERCULOSIS OF THE LARYNX*

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The subject of the treatment of tuberculosis of the larynx has in the past been a favourite one at Annual Meetings of the British Medical Association. It was discussed at Manchester in 1929, at Birmingham in 1911, at Leicester in 1905, and at Cheltenham in 1901, and I have no doubt that there have been other discussions of the subject which I have overlooked. The reasons are not far to seek. The subject is one which interests not only the laryngologist, but the physician (particularly if he specializes in diseases of the chest), the tuberculosis officer, the medical officer of health, and last but by no means least, the general practitioner; for the disease is a common one—tuberculosis is the commonest of all specific infections of the larynx—and the last word on its treatment has yet to be said.

According to the last official report[1] the deaths from tuberculosis of the respiratory system in England and Wales in 1931 amounted to 29,658 (in 1921 it was 33,505, and there is apparently a persistent decline in the mortality). The total number of cases in England and Wales of pulmonary tuberculosis on December 31st, 1931, on the registers of notifications kept by medical officers of health, as shown in the returns, was 236,758

* Read in opening a discussion in the Section of Laryngology at the Annual Meeting of the British Medical Association, Dublin, 1933.